1. Attacks of migraine occur associated with nausea and vomiting; this form is known as simple migraine, and usually remains unaltered during the life of the patient.

2. In other cases visual disturbances (hemianopsia, scintillating scotoma, amaurosis, etc.) are associated with the migraine, and the disease is then known as ophthalmic migraine.

3. When paralysis of the ocular muscles occurs with the migraine the disease is described as ophthalmoplegic migraine.

4. Migraine, especially the ophthalmic form, is related to epilepsy, and the attacks of migraine may precede for many years the convulsive attacks of epilepsy, although in most cases of migraine no convulsions are ever detected.

5. In some cases epilepsy appears in the form of one or more of the disturbances seen occasionally with migraine, and later, even after many years, convulsions develop. The disease may be epilepsy from the beginning. It matters little, with our uncertain knowledge of the pathology of the two diseases, whether we regard these as abortive cases (*formes frustes*) of migraine that later become associated with epilepsy, or as abortive forms of epilepsy (sensory epilepsy), in which the convulsions later become apparent, provided we recognize a relation between some forms of migraine and epilepsy.

FACIAL SPASM AND ITS RELATION TO ERRORS OF REFRACTION.[1]

By E. W. Stevens, M.D.,
OF DENVER, COLO.

Under the general designations "facial spasm," "mimic spasm," "habit spasm," "histrionic spasm," "painless tic," "convulsive tic," and "palmus" are included several groups of cases presenting the common clinical feature of spasm of the muscles supplied by the seventh or facial nerve.

The following groups of cases are recognized by Osler:[2]

1. The secondary form following paralysis, and consisting in spontaneous clonic twitchings recurring at irregular intervals in the paralyzed muscles.

2. Cases due to the irritation of an organic brain lesion either of the cortex or of pressure on the facial nerve from an aneurism or tumor.

3. In many cases no cause can be assigned—the idiopathic form of Gowers.

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[1] Read before the Colorado State Medical Society, June 20, 1899.

[2] American Text-book of the Theory and Practice of Medicine, vol. i. p. 832.

4. Cases which appear to have a reflex origin and which are associated with irritation in branches of the fifth nerve, as in eye-strain, nasal affections, and diseased teeth. This form of spasm is frequently found in children, and while it may be limited to the facial muscles, is also met with in other groups of muscles. This form of spasm has been called "habit spasm" by Gowers[1] and "habit chorea" by S. Weir Mitchell.[2]

Lastly, facial spasm may form a part of the affection described by the French as Gilles de la Tourette's disease, which is characterized by involuntary spasmodic jerkings in certain muscle groups, particularly those of the face, the explosive utterances of obscene words, and fixed ideas.

The symptom-picture differs but little in all forms of facial spasm. The patient has lost control over some of his facial muscles either on one side or both. The spasm is, as a rule, unilateral at first, and may continue throughout to be unilateral, or may become bilateral after a number of years. They are usually of a rapid, electric-like character. There are often a series of quick, quivering contractions. The spasm is usually clonic. If there is tonic spasm there is almost always clonic spasm as well. In the most common form, the idiopathic, the spasm usually attacks one or two muscles at first, and these at infrequent intervals. The patient or his friends may observe a slight twitching of the orbicularis palpebrarum or of the levator anguli oris. These twitchings become more pronounced, occur more frequently, and may spread to neighboring groups of muscles, and after a time a marked admixture of tonic and clonic spasms is usually observed. Voluntary motion in the muscles is perfectly normal, except for the disturbing influence of the spasms. All sensory disturbances are also wanting. There is neither anæsthesia nor pain. At the beginning the muscles most frequently affected are the orbicularis palpebrarum and the zygomatics. Blepharospasm is a spasm—usually clonic—of the orbicularis palpebrarum muscles, manifesting itself usually by an uncontrollable winking or blinking of the eyelids, the so-called nictitating spasm. A more serious and uncomfortable phase of the difficulty is cramp of the entire muscle when the eyelids are closed tightly and violently, so as to be painful to the patient. In the latter tonic type the lids may remain tightly shut for some minutes, days, or even months, and the patient rendered practically blind from inability to use his eyes. Blindness has occasionally resulted in these cases.

Blepharospasm may be caused by an inverted eyelash, a foreign body producing peripheral irritation, or a corneal ulcer; but we are considering here only that form for which no cause can be found in the condition of the eye or its coverings. The most intractable cases of facial spasm are of this type. Blepharospasm may be due to irritation of the ophthalmic branch of the fifth nerve, owing to conjunctivitis, refractive errors, and muscular insufficiencies. Often the irritation is reflected from the nose, the teeth, or the pharynx. Dr. Pershing[1] has recorded a case in which he attributed the cause of blepharospasm to prolonged tension of the nervous system. Some cases are of central or even cortical origin. A lesion, however, would have to be very small indeed so to affect the cortex or subcortex as to cause blepharospasm without affecting any other muscles of the face. The so-called functional cases of blepharospasm often obstinately resist treatment. They may be temporarily relieved by pressure upon the branches of the fifth nerve, which come from the orbit, or upon the branches of the facial, which supply the muscles affected. Sometimes these pressure points, which were first carefully described by von Graefe, are found on the vertebral column.

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[1] Diseases of the Nervous System, 2d edition, vol. ii. p, 633.

[2] Lectures on Diseases of the Nervous System, Lecture VIII.

Facial spasm is usually strictly confined to the distribution of the facial nerve, but occasionally muscles supplied by other nerves may be affected, showing that the spasms are due to some widely distributed organic lesion, affecting, it may be, the trigeminus, vagus, or other nerves.

Spasms of the muscles supplied by the motor division of the trigeminus may exist independently of the facial—the masticatory spasm of Romberg—and may be either tonic or clonic. Cases of clonic masticatory spasm are seen in which the spasmodic movements are frequently repeated, causing a disorder sometimes spoken of as "chattering jaw." The chattering is almost continuous, except when the patient is asleep. It is usually a sign of senility. Trismus, in its narrowest sense, is a tonic spasm of the muscles supplied by the motor branch of the trigeminus, and especially of the masseters, in consequence of which the jaws are held tightly closed.

In neuralgia of the fifth nerve, or tic douloureux, reflex spasm may occur in some of the facial muscles; but we are dealing in this paper with only the painless forms of spasms.

Children frequently, and adults sometimes, present spasmodic movements that have a half-voluntary aspect, but which the patients are unable to control. An eye may be rapidly winked, or the eyeball rolled rapidly upward, or the brow wrinkled, or the facial muscles contorted, or it may be the shoulder is shrugged or a forward movement of the head or jaw is made. A peculiar fidgety movement of the head or arm is common; more rarely leg movements occur. In other cases the respiratory muscles are involved and various disordered sounds are emitted by the patient. This group of symptoms has been described by S. Weir Mitchell[1] under the term "habit chorea." Gowers,[2] however, considers the name a misnomer, and, believing the affection to be a form of spasm or tic, has given it the name of "habit spasm." This affection usually begins between the ages of six and fourteen years. Rarely it begins late in life, and is then generally permanent. When it commences in childhood the spasms usually cease after a few months or a few years. In adults in whom the disorder has lasted for some time it is difficult, if not impossible, to arrest the movements.

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[1] American Medico-Surgical Bulletin, June, 1895.

Often habit spasm arises from nasal diseases or diseases of the rhinopharynx. In the greatest number of cases, however, some ocular defect, as an error of refraction or insufficiency of the muscles, is responsible for the affection, as has been well shown by Wharton Sinkler[3] and G. E. de Schweinitz.[4]

The etiology of facial spasm is often most obscure, the most common type being separated into a class called idiopathic. The origin of the disease is cleared up during the life of the patient only if, after the removal of some primary cause, the spasms cease. Many cases are undoubtedly of reflex origin, and especially irritation of the fifth nerve, to which the facial has a special reflex relation.

Hirt[5] reports a case of blepharospasm, which had persisted for years and was considered hopeless, which was cured after a flexion of the uterus had been improved. Bettremieu[6] has recorded three cases of facial spasm due to irritation of the filaments of the fifth nerve in the lachrymo-nasal canal.

Many cases of cortical spasm have been recorded. One of the earliest and most remarkable of these is the case recorded by Berkley and quoted by Mills. In this case the spasm was confined to the zygomatics of the left side of the face. On autopsy a nodule of calcareous degeneration was found in the precentral convolution of the right side, about one and a half inch above the Sylvian fissure.

Facial spasm is frequently the signal symptom in cortical or subcortical disease. It may be produced by a small, gross lesion anywhere from the facial muscles to the peripheral distribution of the nerve. Cases have been reported in which tumors, aneurisms, or foci of softening or degeneration have produced facial spasm. According to Mills,[7] in all these cases due to coarse lesion, whether cortical, subcortical, nuclear, or neural, the spasm is coarser and more irregular in type than in other cases.

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[1] Loc. cit.

[2] Loc. cit.

[3] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, May, 1897.

[4] Proceedings of Philadelphia County Medical Society, 1888, p. 144.

[5] Diseases of the Nervous System, Hock's trans., p. 81.

[6] Archives d'Ophthalmologie, April, 1899, p. 246.

[7] The Nervous System and its Diseases, p. 922.

A number of cases of facial spasm have come under my observation in the past few years. In some of these cases an examination and treatment of the eyes proved of distinct advantage to the patient; in others the results were negative so far as influencing the spasms was concerned.

Case I.—F. E., aged twenty-two years; occupation, barber. General health good. No history of rheumatism or chorea. For the past ten months had complained of eyes closing and shutting rapidly, with a quick, snapping movement, which had compelled him to abandon his occupation. No other muscles affected. An examination of his eyes gave the following results: O. D., 4/6; O. S., 4/6. Esophoria, 7 centrads. The external anterior segments of the eyes were normal, and there were no notable changes in the fundus. There was distinct dread of light. Atropine was ordered and continued for about a week. Under this mydriatic the refraction was found to be O. D. + 0.50 S. = 1.00 C. ax. 60°. O. S. + 1.50 C. ax. 120°.

These glasses were ordered for constant wear. Constitutional treatment was given in the form of Fowler's solution of arsenic in ascending doses. In four months the blepharospasm had entirely disappeared.

Case II.—A. B., aged forty-four years, a carpenter by occupation, applied for treatment at Dr. Edward Jackson's service at the Wills Eye Hospital, Philadelphia. This patient was of fine physical development, with no personal or family nervous history. For the past ten years had had severe bilateral blepharospasm of the clonico-tonic type. For several years had been unable to attend to work on account of the violence of the twitchings. This case had been under the care of several competent ophthalmologists and neurologists, and had taken long courses of arsenic, galvanism, and antispasmodic remedies, without avail. Both facial nerves had been stretched, with recurrence of spasms in a few months. The examination of his eyes gave the following results: O. D., 4/8; O. S., 4/8. The conjunctiva was injected, but not catarrhal. Small oval disks, with retinal striations above and below. Under duboisine the refraction was found to be O. D. + 1.00 S. = + 1.50 C. ax. 90°. O. S. + 1.50 S. = + 1.00 C. ax. 90°

The full correction was ordered, but no improvement had followed when the patient removed from the city, a couple of months later.

Case III.—Mrs. Mary G., aged forty years; in fair general health. When twenty-four years of age she first noticed twitching of the lids of both eyes, which has slowly grown worse, in spite of treatment. For years she has been unable to read or use her eyes for near work. She now presented violent clonic, bilateral blepharospasm, with involvement of the zygomatics. The eyes were as follows: O. D. and O. S., 4/4; amplitude of accommodation, 4 D.; esophoria, 3 centrads. Anterior segments and fundi normal in all respects, and under atropine mydriasis the patient was found to be wearing the right correction, + 1.00 D. in each eye.

Both facial nerves were now stretched under ether, and the stretching was repeated two days later, as very little influence had been produced upon the spasms by the first operation. The second vigorous stretching caused facial paralysis, with, of course, entire cessation of the spasms. Unfortunately, with the return of voluntary power in the muscles in six weeks, the spasms also returned with their former force and frequency.

Case IV.—Miss B. S., aged seventeen years, a remarkably healthy-looking, well-nourished girl. This patient was of a highly excitable, nervous temperament, inherited from her father. For the past six months she had suffered from rapid blinking and shutting of her eyes, jerkings of her head, and at times twitchings of her arms and legs During this period she was under the professional care of a well-known neurologist.

An examination of the eyes had been made, but owing to the lack of control of the patient it was regarded as imperfect and unsatisfactory. My examination yielded the following results: O. D., 4/30; O. S., 4/20. Lids, lashes, and conjunctiva were normal and cornea clear. It was beyond my power to test the muscle balance or obtain a satisfactory study of the fundus oculi, on account of the jerkings of the patient and the marked dread of light.

Atropine mydriasis was produced and kept up about ten days. Under this the refraction by the shadow-test was found to be O. D. —1.00 S. = —2.00 Cyl. ax. 30°. O. S. = —1.50 —1.50 Cyl. ax. 180°. A full correction was ordered, and arsenic was once more given in ascending doses. One month after the spasms had nearly subsided, and at the end of two months they had entirely ceased. There had been no return of spasms two years after, when I last saw her.

Case V.—Mrs. H. R. H., aged thirty-six years, referred to me by Dr. Jameson, of Philadelphia. This patient was a finely developed woman in perfect health. Ten years ago she first noticed twitchings of the lids of the left eye, which after a few months were accompanied by twitchings of the muscles of the face on the same side, and gradually grew worse. During this time she had consulted many of the most eminent neurologists, both in this country and abroad, being for several months under the care of the late Prof. Charcot. Strange to record, during all this treatment by electricity, arsenic, and antispasmodics her eyes had never been examined. The examination of her eyes now showed that they were normal in all respects with the exception of a refractive error of + 0.50 D. in each eye. These glasses were ordered for all near work, but, as was expected, without the slightest influence upon the force or frequency of the spasms.

Case VI.—Alice C., aged eight years. For the past two months had had persistent spasmodic bilateral winking, compelling her withdrawal from school. No history of rheumatism; no twitchings of the muscles of the face or jerkings of the limbs or head The eye examination was as follows: O. D. and O. S., 4/6. Muscle balance not taken. Refraction under atropine gave O. D. + 1.00 Cyl ax 90°. A S + 1.00 Cyl. ax. 90°. This was ordered for constant wear, and arsenic given in increasing doses. Six weeks later the winking was entirely cured.

These cases illustrate the importance, in the treatment of facial spasm, of fully correcting the error of refraction in conjunction with proper internal medication. It is essential in every case to determine the existence of the cause and remove it.

It is not my intention to attribute to errors of refraction an undue importance as exciting agents in the causation of facial spasm. A large number of cases may be strictly traced to diseases of the pharynx and nasal diseases, as has been pointed out by Jacobi,[1] and in a still larger group no cause can be assigned.

I wish to emphasize the following points:

1. The refraction and muscle balance should be carefully examined.
2. The refraction should be determined under complete mydriasis and the full correction ordered.
3 It is not sufficient to get rid of the cause, for the nerve-centres, having acquired a vicious habit, do not recover their normal condition until systematic treatment has been pursued.

CYSTINURIA AND ITS RELATION TO DIAMINURIA.

By Charles E. Simon, M.D.,
OF BALTIMORE, MD.

Cystin, which was first discovered by Wollaston in 1805, in a vesical calculus obtained from a child five years of age, is seen only in the urine under conditions which are essentially pathological in their nature. It may occur in solution or as a simple deposit, and thus be eliminated without giving rise to any disturbance. More commonly, however, concretions are formed in the kidneys or bladder, leading to attacks of renal colic and to symptoms of vesical irritation. It has been argued that as these cases only are seen by the physician, cystinuria may be much more common than is generally supposed, and this is no doubt true to a certain extent; but if we remember the care with which the urine of practically every patient is examined at so many of the leading hospitals of the world, and compare with the total number of such examinations the actual number of cases of cystinuria recorded in the literature, it is apparent that the condition is nevertheless one of great rarity.

During the last ten years I have examined not less than fifteen thousand specimens of urine, and the case of cystinuria presently to be described is the only one that has come under my immediate observation. At the Johns Hopkins Hospital and Dispensary only three cases have been seen during the same period of time.

The first case of cystinuria, as I have already indicated, was described by Wollaston, who recorded his discovery, together with the history of a second case, in 1810. Niemann, in his excellent monograph, published in 1876, gives an account of fifty-two cases which he has collected from the literature, and adds a further case. I have arranged these in chronological order in the table below, and have attempted to complete the list to the present time.

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[1] The American Journal of the Medical Sciences, 1896.